AMENDED SPECIFICATION

Example 4

Construction of RADSs to prevent *Streptococcus equi* infection and disease.

Streptococcus equi causes a very severe disease of racehorses and other equines called strangles (Nara, P. et al. (1983) Am. J. Vet. Res. 44:529-534). Even worse is the immune complex disease *Purpura hemorrhagica*, a consequence of earlier in life *S. equi* infection (Galan, J. and J. Timoney (1985) J. Immunology 135:3134-3137). *S. equi,* like group A streptococci, produce an M protein on the surface which is antiphagocytic and thus a major virulence determinant enhancing the success of *S. equi* infection. Antibodies to the *S. equi* M protein (SeM) are opsonic and facilitate the successful phagocytosis and killing of *S. equi* (Galan, J. and J. Timoney (1985) Infect. Immun. 47:623-628). Thus an antibody response to SeM is likely to be highly protective in preventing *S. equi* infection of horses. The RAV pMEG-573 encoding the *S. equi* SeM protein is depicted in Figure 18. This RAV was obtained by cloning the PCR fragment flanked by primers SeM444-474 GCGAACTCTGAGGTTAGTCGTACGGCGACTC (SEQ ID NO:1) and SeM1265-1233 TTGATCAATTTCTGCTAATTTTTGAGCCATTTC (SEQ ID NO:2), containing the central portion of the SeM coding region from the SeM clone pSEM06, into the *Nco*I and *Bam*HI sites of pMEG-546. pMEG-573 is only dependent on the presence of the Δ*ilvG3*::TT*araC*P_{BAD}*lacI*TT deletion/insertion mutation (Figure 2) in the chromosome to repress the runaway phenotype and SeM expression. The vaccine strains for SeM also contain either the Δ*phoP1918* or Δ*phoP24* attenuating deletion mutation (mutations 8 and 9, Figure 2). A comparison of the level of SeM expression by different attenuated *Salmonella* vaccine strains, in which SeM